Affective Responses to Laboratory Stressors in Rheumatoid Arthritis Patients

A Comparison of Mindfulness-based Emotion Regulation and Cognitive Behavioral Interventions

by

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ABSTRACT

This study examined whether cognitive behavioral therapy and mindfulness interventions affect positive (PA) and negative affect (NA) reports for patients with rheumatoid arthritis (RA) before, during, and after stress induction. The study also investigated the effects of a history of recurrent depression on intervention effects and testing effects due to the Solomon-6 study design utilized. The 144 RA patients were assessed for a history of major depressive episodes by diagnostic interview and half of the participants completed a laboratory study before the intervention began. The RA patients were randomly assigned to 1 of 3 treatments: cognitive behavioral therapy for pain (P), mindfulness meditation and emotion regulation therapy (M), or education only attention control group (E). Upon completion of the intervention, 128 of the RA patients participated in a laboratory session designed to induce stress in which they were asked to report on their PA and NA throughout the laboratory study. Patients in the M group exhibited dampened negative and positive affective reactivity to stress, and sustained PA at recovery, compared to the P and E groups. PA increased in response to induced stress for all groups, indicating an "emotional immune response." History of recurrent depression increased negative affective reactivity, but did not predict reports of PA. RA patients who underwent a pre-intervention laboratory study showed less reactivity to stressors for both NA and PA during the post-intervention laboratory study. The M intervention demonstrated dampened emotional reactions to stress and lessened loss of PA after stress induction, displaying active emotion regulation in comparison to the other groups. These findings provide additional information about the effects of mindfulness on the dynamics of affect and adaptation to stress in chronic pain patients.

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Chapter 1

INTRODUCTION

The stress response is a normal reaction to stimuli in the environment. Our reactions to potentially harmful stressors are adaptive, and allow us to engage in self-preservation behaviors. In the face of environmental or physiological challenge, activation of the autonomic sympathetic nervous system prepares the organism to engage in these self-preservation behaviors, while activation of the parasympathetic nervous system returns the organism's systems to homeostasis. These systems work in tandem. A life of chronic stress, such as that seen in chronic illness, can disrupt the equalizing tendency of these opponent processes and contribute to poorer mental and physical health outcomes as well as make it difficult to sustain a positive, meaningful life (Davis, Zautra, & Smith, 2004). Individuals with rheumatoid arthritis (RA); a chronic, systemic, and progressive autoimmune disease characterized by inflammation of the joint lining and chronic pain; endure daily stresses along with the pain and uncertainty of living with a chronic ailment.

An examination of these daily experiences allows us to gain a clearer picture of how the ebb and flow of stress relate to the physical health of an individual with RA. In a review of 27 independent studies stress from minor life events was related to increased disease activity (Herrmann, Scholmerich, & Straub, 2000). Zautra and colleagues (1997) found that for RA patients experiencing a stressful episode, increases in the number of stressful interpersonal events in one week were associated with increased patient-reported joint tenderness, clinician's global rating of disease activity, and increased immune activity in the same and subsequent weeks. Interleukin-6 (IL-6), a proinflammatory cytokine associated with the acute inflammatory response, has been implicated as a potential mediator for RA disease activity. Elevated circulating levels of IL-6, indicative of systemic inflammation, have been found in RA patients undergoing stressful circumstances: chronic interpersonal stress recorded daily over a period of 30 days was related to increases in stimulated IL-6 (Zautra et al., 2008; Davis et al., 2008). Over time, prolonged increases in disease activity can contribute to disease progression. Thus, management of stress is of utmost importance for patients with RA. For individuals with RA, daily stresses go beyond the

usual psychological distress. They have the potential to disrupt physiological processes and impact physical functioning.

To manage stress and pain, the standard behavioral treatment for RA is pain management. RA is an autoimmune disease of unknown etiology that can severely limit physical functioning, increase disability, and cause severe pain (National Institutes of Health, 2009). There is no cure—behavioral treatments are implemented with the aim to instruct RA patients in specific strategies to cope with pain as it arises. Cognitive behavioral therapy (CBT) is the standard behavioral treatment and randomized clinical trials comparing psychosocial therapies have shown CBT to be especially effective in improving pain coping, while also reducing pain, and mitigating depressive symptoms to a lesser degree (Astin, Beckner, Soeken, Hochberg, & Berman, 2002). Reduction of pain is a worthwhile and valued endeavor; however, these pain management interventions sometimes neglect to directly target the psychological distress that results from and contributes to chronic pain.

For RA patients, pain is a part of life and the chronicity of this syndrome provides ample opportunity for management of stress-related pain. Pain management is obviously a priority in management of this chronic disease; however, interventions that encourage better emotion regulation by increasing the ability to regulate negative affect (NA) and the ability to view one's happiness independent of external and potentially discouraging circumstances can have the added benefit of improving psychological well-being. Adopting this mindset may be better in the long run because over time, it promotes better strategies to deal with stress, instead of coping with pain as it arises.

Mindfulness-based interventions are aimed at these goals. Definitions of mindfulness vary, but generally include an attentional component and an attitudinal, acceptance-based component (Bishop, 2004). Mindfulness has been found to predict less negative psychological distress and increased psychological flourishing, here relayed as two related but independent processes (Reich, Zautra, & Davis, 2003). Some of the proposed mechanisms that explain the relationship between mindfulness and mental health are: clarity about one's internal experience,

the ability to regulate NA, and the ability to view one's happiness independent of external circumstances, rumination (Coffey, Hartman, & Fredrickson, 2010).

Zautra and colleagues (2008) tested a mindfulness-based intervention (M) targeting affective disturbance in RA patients. Mindfulness-based programs have been implemented in various clinical populations with much success (Baer, 2003). Teasdale and colleagues (2000) implemented mindfulness-based cognitive therapy (MBCT) to prevent recurrence of major depression by reducing metacognitive awareness and changing the individual's relationship to negative thoughts versus changing thought content (Teasdale et al., 2002). The intent to change experience, rather than behaviors, is consistent in mindfulness-based stress reduction (MBSR) for individuals with chronic pain (Kabat-Zinn, 1982; McCracken, Gauntless-Gilbert, & Vowles, 2007). Beyond blunting the negative impact of chronic pain in reducing perceptions of bodily pain, improving physical functioning, decreasing occurrence of psychiatric symptoms, and lessening perceived stress (e.g., Rosenzweitg et al., 2010; Shapiro, Oman, Thoresen, Plante, & Flinders, 2008; Carmody & Baer, 2008), mindfulness-based interventions also promote well-being (Brown, Ryan, & Cresswell, 2007). In a cohort study examining the effects of MBSR in chronic pain patients, individuals with arthritis reported pre to post changes in increased perceptions of health (d = .82) and increased vitality (d = .88) (Rosenzweig et al., 2010).

The current investigation is based on Zautra and colleagues' research on emotion regulation and adaptation to chronic pain (e.g. Zautra, Smith, Affleck, & Tennen, 2001; Zautra et al., 1995; Zautra, Affleck, Tennen, Reich, & Davis, 2005). Research from this lab and others suggests positive interpersonal events promote resilient functioning and positive well-being (Zautra et al., 2008). Utilizing a resilience-based approach, the M intervention's intent was twofold: to reduce the negative impact of stressful life events, and to enhance the ability to sustain positive social engagements in spite of pain and stress.

In a sense, both M and P interventions target the stress response. The P intervention solely aims to manage pain and in doing so it may reduce stress associated with a pain episode, or perhaps alleviate existing stress exacerbated by pain. While P intervention strategies are reactionary to the stressor and may aid in recovery from stress related to pain, the M intervention's

focus on more effective emotion regulation may actually dampen the initial stress response. Mindfulness training promotes non-reactive awareness to negative emotions and thoughts, resulting in a "distancing" from these potentially reactive states (Chambers, Lo, & Alle, 2008). This mechanism may explain why RA patients in the M group have lower average levels of NA and higher levels of PA. These individuals may continue to feel the same levels of pain, but they experience less distress about the pain. It can be extrapolated that RA patients in the M condition were less reactive to stress. The use of self-report diary data is useful in describing the interplay between stress and affect. However to more definitively examine differences in stress response, examination of stress responses within a laboratory stress induction paradigm is needed.

The current study extended Zautra and colleagues' (2008) mindfulness intervention research by examining responses to induced stress within a controlled, laboratory setting. To the author's knowledge, no studies have utilized stress induction to examine treatment effects in a mindfulness-based intervention. Careful examination of the stress response is particularly important in research in RA, a disease characterized by stress reactivity. Because the M condition focused on emotion regulation, measures of affective reactivity and recovery from induced stressors allowed for the evaluation of the intervention's proposed mechanism of change. Emotional reactions to stress have the ability to perpetuate and worsen the effects of a stressor (Chida & Hamer, 2008). Targeting affective responses to stress may mitigate the negative effects of stress on an individual and promote emotional well-being over and above that which may occur through cognitive pain management alone. Thus, this study of affective reactivity furthers our understanding of how individuals emotionally respond to stress, and whether an emotion regulation intervention (M) can directly affect and potentially change the stress response.

As we consider the role of affective response to stress, we must also consider the role of underlying affective disturbance. Several studies report an association between arthritis and psychiatric disorders—depression in particular. In a review of 12 independent studies comparing depression in RA patients and depression in healthy controls, rates of depression were higher in RA patients than in the controls (Dickens, McGowan, Clark-Carter, & Creed, 2002). The direction of the relationship between depression and arthritis was further clarified in Land and

colleagues' (2010) population-based longitudinal study in which arthritis diagnosis predicted later depression. In response to Land and colleagues, Nicassio (2010) added that while pre-existing depression did not predict the onset of arthritis, secondary depression contributed to adverse health outcomes such as interference of functioning, reduction of medical adherence, and use of maladaptive health behaviors. Additionally, depression exacerbates the inflammation process, and this has been supported in studies showing increased levels of proinflammatory cytokines in depressed individuals with RA, compared to non-depressed patients (Zautra, Hamilton, Potter, & Smith, 1999; Zautra et al., 2004).

The parent study from which this current investigation is based has already yielded valuable information about the relationship between stress and recurrent depression in RA patients. During stress induction, RA patients with a history of two or more episodes of major depression reported more pain at baseline, and higher pain in response to stress induction compared to RA patients with one episode or no history of depression (Zautra et al., 2007). Interestingly, the two groups did not differ in average pain and other daily diary measures, but patients with a history of depression had significantly stronger associations between pain and various aspects of daily emotional experience than did the never-depressed patients (Zautra et al., 2006). The previously depressed also engaged in pain coping by venting emotions, reported higher negative mood, and lower positive mood—even after controlling for current depressive symptoms (Zautra et al., 2008). These findings indicated that recurrent depression was associated with greater pain and pain reactivity, and more mood disturbance, suggesting the presence of emotion dysregulation.

RA patients with a history of recurrent depression represent a subset of RA patients particularly sensitive to emotional turmoil related to pain and stress. Patients in the M intervention with a history of recurrent depression benefited the most in affective functioning. These RA patients reported significant changes from pre-intervention to post-intervention in decreases in NA and increases in positive affect (PA) relative to patients in the P and education only control groups (Zautra et al., 2008). This finding suggests better daily emotion regulation in the M group, especially for RA patients with a history of recurrent depression. In effect, these

patients were better able to capitalize upon the emotion regulation skills learned in the M group relative to the other groups. It was expected that these group differences would be reflected similarly under conditions of laboratory-controlled stress induction.

The current investigation primarily examined group differences in affective response to lab-induced stressors. The outcomes of interest were negative affect (NA) and positive affect (PA). The main study hypotheses were as followed:

1. All groups (M, P, E) would demonstrate quadratic functions across the lab induction procedure.
    a. For all groups, NA would demonstrate a negative quadratic function, with NA increasing and peaking during implementation of the stressors, then decreasing back to baseline levels of NA once stress induction is complete.
    b. All groups (M, P, E) would demonstrate a positive quadratic function of PA across the lab procedure, with PA decreasing during stressors, and increasing back to baseline levels of PA once stress induction was complete.
2. All groups would exhibit similar changes in NA and PA after initial presentation of the stressor—there would be no group differences in initial reactions to stressor. The M group would exhibit quicker affective recovery relative to P and E groups, and this would be evident in
    a. Lower NA, after initial stressor (a more negative linear function)
    b. Higher PA after initial stressor (a more positive linear function)

Secondly, the potential effects of a history of recurrent depression (RD+) were explored to evaluate whether this subset of individuals differed from RA patients without a history of recurrent depression (RD-)

3. It was predicted that M/RD+ would demonstrate faster recovery than all other groups, as evidenced by the greatest:
    a. Decreases in NA after initial stressor was presented, and
    b. Increases in PA after initial stressor was presented.

Lastly, potential testing effects of the Solomon-6 design utilized in the study were explored. Approximately half of the participants underwent a pre-intervention laboratory session.

4. It was predicted that testing effects were likely to be evident, with participants who underwent a pre-intervention lab being less reactive to the stressors at post-intervention lab stress induction. The same affective patterns would emerge, but to a lesser degree.

Chapter 2

METHOD

Overview of the Study

Once screened into the study and consented, participants completed initial questionnaires about demographic information. Participants were then clinically evaluated for a history of major depression. A second set of questionnaires assessing for pain and depressive symptoms was then completed. Prior to the intervention, participants completed 30 days of daily diaries assessing joint pain, negative and positive affect, and depressive symptoms. Upon completion of the daily diaries, half of participants were randomly selected to undergo a pre-intervention laboratory pain assessment with stress induction. Blood draws were collected at different time points over the course of the lab. Twenty to 28 participants were grouped into one of the eight intervention waves. Participants were randomly assigned to one of three treatment conditions using a random numbers table. At post-intervention, all participants underwent diary assessment and laboratory assessment.

Participants

A total of 144 participants (68.1% women, 31.9% men) were randomized to receive 1 of 3 study interventions. Of the 144 participants, 16 either dropped from the study, did not complete the intervention, or were lost to follow-up. The remaining 128 participants completed a post-intervention laboratory assessment and were included in the analyses. Participants were recruited from the Phoenix, AZ metropolitan area through solicitations at health fairs, to Arthritis Foundation members, local physician offices, and from rheumatologist referrals at the Carl T. Hayden Veterans Affair (VA) Medical Center. Written confirmation of RA diagnosed from rheumatologists was required. RA patients taking cyclical estrogen replacement therapies or with a history of lupus were excluded from the study. The average duration of RA disease was 11.5 years for female participants and 16.1 years for male participants. The mean age was 51.2 years for women and 61.9 years for men. Eighty-five percent of the women and 83% for the men identified as Caucasian. Average annual family income for men was in the $25,000 to $29,000 range and $30,000 to $39,000 range for women.

Intervention Plan

The study compared a mindfulness-based emotion regulation program (M) with a cognitive behavioral pain management (P) program. The interventions were compared to an education control group (E).

All intervention conditions followed an analogous format. Each 8-week treatment was comprised of weekly 2-hour modules with specific themes relating to the content of the intervention. The initial session included an intervention overview and rationale for use. Subsequent sessions addressed specific objectives to build on skills relevant to the intervention. Treatment sessions were facilitated by a doctoral level psychologist and an advanced doctoral student trained in CBT methods and behavioral medicine. Therapists introduced educational information, implemented skill-based exercises, and reviewed participant understanding and application of skills learned. Therapists also assigned weekly homework related to session activities, followed by a review of homework at the beginning of the following session to reinforce learning. Interventions were carried out in groups of 5 to 8 participants (average group size = 6).

Pain management (P). The P intervention utilized standard cognitive behavioral techniques to increase pain management skills. Modules included: (a) introduction of pain concepts; (b) relaxation training; (c) autogenic training and other relaxation techniques; (d) activity pacing and daily activities management; (e) cognitive coping; (f) alternative pain management approaches; memory and concentration; (g) managing intense pain episodes; problem-solving; and (h) relapse prevention, generalization, and maintenance.

Mindfulness-based emotion regulation (M). Unique to other mindfulness-based therapies, this program emphasized skills training in order to sustain positive emotional experience, especially with regard to interpersonal relationships. The M intervention utilized mindfulness meditation components common to other mindfulness-based interventions like MBSR (Kabat-Zinn, 1982) and MBCT (Teasdale et al., 2000), though relatively shortened in duration in order to ensure an equivalent experience across group conditions.

The program modules included: (a) mindfulness and the bidimensional model of emotion; (b) mindfulness and awareness; (c) emotional clarity and well-being; (d) acceptance, negative thoughts, and reframing; (e) positive emotions and pleasant event scheduling; (f) enhancing social relations; (g) intimacy, stress, and mindfulness; and (h) maintenance and generalization.

Education-only (E). The E condition served as a comparison group to the M and P interventions by controlling for nonspecific therapeutic effects. This condition consisted of a series of instructional presentations about the RA condition and related topics in health. Modules in the E condition included: (a) introduction to RA: definitions, pathophysiology, and epidemiology; (b) prognosis and treatment, diagnostic tests, medical specialists; (c) RA medications, medication use; (d) neurophysiology of pain (surgical intervention); (e) natural remedies (nutrition, diet); (f) exercise and sleep; (g) communication with your doctor and traveling with RA; and (h) review and group closure.

The utmost effort was put forth in ensuring the objectives for each condition were addressed only in the corresponding intervention. For instance, stress and pain management were only discussed in relation to sustaining positive well-being within the M intervention. Discussions about emotions and well-being were not addressed in the P group—which solely focused on how to manage pain. Coping strategies and emotion were purposefully omitted in the E group.

Procedure

At the outset of the lab assessment participants were given detailed instructions about the lab procedure, in addition to a second informed consent. An initial 10-minute rest period was used to establish baseline measures prior to periods of stress induction—a standard speech task (Davis, 1999; Davis, Twamley, Hamilton, & Swan, 1999) and a discussion about a recent interpersonal conflict designed to induce stress (Davis, Zautra, & Reich, 2001). A 20-minute recovery period following the stress inductions followed. Self-report measures of PA and NA were taken at baseline, after each stress induction, and after recovery for a total of 4 assessments.

Baseline period. The participant was asked to sit quietly and rest while relaxing music was played. A research assistant suggested to the participant to take a mental vacation to a

favorite place where s/he felt safe and comfortable, while relaxing his or her body and mind. After 10 minutes, a blood sample was collected and self-report questionnaires administered.

First stress induction. Then the participant was informed that s/he would take the next 10 minutes to prepare a 5-minute speech describing his/her best and worst characteristics. The participant was told that two research assistants would be present to evaluate the speech for content, clarity, and style; and that the speech would be taped so that a team of psychologists could evaluate the speech at a later time. If the participant stopped before the 5 minutes were over, s/he was asked to continue. If the participant was unable to continue, the research assistants remained in the room without speaking until the 5 minutes had passed. Once the speech task was complete, the second blood sample and second set of questionnaires was collected.

Second stress induction. For the second stress induction the participant was asked to think about a recent conflict with an important person in his/her life that was stressful and elicited strong feelings at the time. First, the research assistant asked the participant to visualize the event in great detail (i.e. where the conflict took place, what things they could see, hear, and smell). Then the participant was told to attend to the person involved in the conflict—how they entered, what s/he looked like, what was done or said. The research assistant then directed the participant to reflect on his/her own thoughts and physiological reactions (change in heart rate, tension in muscles). Finally, the participant was asked to describe the event to the research assistant paying special attention to emotions and feelings experienced, cognitions, how s/he coped, whether s/he talked about the event to someone else, and what s/he would have done differently. The stress interview continued for 15 minutes, followed by a third blood sample and set of questionnaires.

Recovery period. Lastly, the participant was asked to relax in the same manner as the baseline period. After 20 minutes, the fourth and final blood sample and set of questionnaires was collected. The participant was then debriefed and given $90 for participation.

Measures

Structured clinical interview for DSM IV (SCID-I). The SCID-I was used to assess for history of major depression. Interviews were conducted by advanced clinical psychology students or postdoctoral students trained to administer and code the SCID-I, under the supervision

of a licensed clinical psychologist. Interviews were conducted by telephone and were audiotaped with the participants' consent (see Zautra et al., 2007 for a detailed description of the depression assessment procedure). Telephone interviews are equivalent to face-to-face interviews in assessing Axis I depressive disorders (Rohde, Lewinsohn & Seeley, 1997; Simon, Revicki, & VonKorff, 1993). Major depressive episodes could not be due to uncomplicated bereavement, injury or illness, alcohol or drugs, or medication use. Recurrent history of depression (RD) is defined as two or more episodes of major depression.

Positive and negative affect. PA and NA were measured using the Positive and Negative Affect Schedule (Watson, Clark, & Tellegen, 1988). Participants were given a list of 10 positive mood adjectives (current sample *alpha* reliability = .906) and 9 negative mood adjectives (*alpha* = .879). The NA subscale of the PANAS is comprised of 10 negative mood adjectives, however 1 negative mood adjective was left out in error, thus a modified 9-item measure of NA was assessed instead. Participants were asked to rate the extent to which they felt these mood adjectives at that point in time using a 5-point scale from 1 ("very slightly or not at all") to 5 ("extremely"). PA and NA were assessed at four time points during the experimental session.

Analytic Strategy

This study employed several sets of analyses utilizing multilevel random effects modeling. The data were coded by Lab Interval, a repeated, within person factor with four levels that corresponded with the four lab intervals during the stress induction procedure: baseline, stressor 1, stressor 2, and recovery. Linear and quadratic effects of Lab Interval were expected and modeled accordingly. The interactions of Lab Interval by Treatment Group were indicative of differential effects of the interventions over the duration of the laboratory stress induction session. The first set of analyses looked at differences in affective outcome for all participants who completed a post-intervention laboratory session. In light of the correlation between PA and NA, a subset of these analyses included PA as a covariate when testing for differences in NA, and conversely NA as a covariate for analyses with PA as the outcome. The second set of analyses explored the effects of a history of recurrent depression on study outcomes. The third and final set

of analyses explored testing effects on PA and NA resulting from the Solomon 6 design of the study.

The outcomes investigated were post-intervention PA and NA. The independent variables included in the models were: assigned Treatment Group and Lab Interval. Treatment Group was treated as a categorical between person factor with three levels: education control, pain management, and mindfulness/emotion regulation. Lab interval was a repeated, within person factor with four levels that correspond with the four lab intervals during the stress induction procedure: baseline, stressor 1, stressor 2, and recovery. For each lab assessment, self-reports of NA and PA were collected at each of the four lab intervals. Categorical predictor variable Pre/Post delineated between participants who received both pre- and post-intervention lab assessments and participants who received post-intervention lab assessments only. Significant interactions with Pre/Post indicated the presence of testing effects introduced by the Solomon 6 study design. History of Recurrent Depression was treated as a dichotomous variable, where RD- represented individuals with a history of 0 or 1 major depressive episode, and RD+ represented those with a history of 2 or more major depressive episodes.

All multilevel analyses were conducted using SAS PROC MIXED (Littell, Milliken, Strong, & Wolfinger, 1996). The MIXED procedure utilized estimation techniques that allowed for missing data due to data collection errors and/or planned missing observations, thus all 128 participants were included in the analyses. Treatment effects, testing effects, and effects of recurrent depression were estimated with dummy coded variables. For example, the basic equation to assess NA would be as follows:

Negative affect = b_0 + b_1 Lab Interval (linear) + b_2 Lab Interval (quadratic) + b_3 Treatment Group + b_4 Lab Interval (linear) x Treatment Group + b_5 Lab Interval (quadratic) x Treatment Group + residual error

To assess for the effect of recurrent depression, Depression History was included in the basic equation above and allowed to interact with the other predictors. To assess testing effects, Pre/Post was included in the equation above and allowed to interact with the other predictors. PA was modeled in the same manner as NA.

An ARH (1) error structure was used to model significant heterogeneity in the autocorrelation between adjacent Lab Interval scores within person. An unstructured covariance matrix was selected for models that did not have significant autocorrelation between Lab Interval scores.

Chapter 3

RESULTS

Preliminary Comparisons of Groups

Table 1 displays the demographic characteristics of the study sample across the three intervention groups. The treatment groups were comparable in age ($F(2,127) = 1.45$, $p = .24$), gender ($\chi^2(2, N = 128) = 5.39$, $p = .07$), ethnicity ($\chi^2(8, N = 125) = 8.45$, $p = .39$), and duration of time from RA diagnosis to study entry ($F(2,123) = 1.34$, $p = .27$). History of recurrent depression did not differ between treatment groups ($\chi^2(2, N = 127) = 3.02$, $p = .22$). The number of sessions attended did not vary across groups ($F(2,94) = 2.13$, $p = .13$), indicating comparable "dose" of intervention received across treatment groups.

Group Effects on Negative Affect

Two sets of analyses were required to assess for all possible group comparisons. The first analysis contrasted intervention groups P and M against attention control group E. The second analysis was a direct comparison of group P versus group M. For all comparisons, significant linear and quadratic effects (all p values < .0001; see Tables 3 & 4) were present for NA across the four intervals during the stress induction procedure. NA increased from baseline to stressor 1, NA continued to increase from stressor 1 to stressor 2, and finally NA decreased from stressor 2 to relaxation (see Figure 1), supporting Hypothesis 1a.

Group comparison analyses indicated the M group was significantly different than P and E groups (see Tables 3 & 4). P and E groups did not differ significantly. Significant Group effects showed that the M group reported lower levels of NA overall ($M_M = 1.33$, $SD_M = .53$) than the E group ($M_E = 1.52$, $SD_E = .67$, $t = 2.01$, $p = .047$) and the P group ($M_P = 1.54$, $SD_P = .74$, $t = 1.78$, $p = .079$; see Table 2 for means and standard deviations). Significant 'Group x Interval' linear and quadratic effects (all $ps < .05$) also demonstrated that M was statistically different than P and E groups.

Separate analyses were conducted for each Group to obtain slope coefficients to allow for comparison between groups (see Table 5). The M group demonstrated a shallower quadratic 'Group x Interval' effect than the E and P groups ($ß_M = -.23$, $ß_E = -.35$, $ß_P = -.36$).

Covarying positive affect on NA. PA was included as a covariate in the model to assess for NA independent of PA. Analyses of NA controlling for PA yielded comparable results to analyses in which PA was not included as a covariate (see Tables 6 & 7 for group comparisons and Table 8 for beta coefficients). Three of four fit statistics indicated a worsening of goodness of fit with the inclusion of PA as a covariate. Because covarying PA did not improve goodness of fit, the use of PA as a covariate was not included in subsequent analyses of NA.

History of recurrent depression on NA. The effects of a history of recurrent depression were assessed by including RD as a predictor for NA. Again, two sets of analyses were conducted to assess for all possible group comparisons (see Tables 9 and 10). Significant group differences were only evident between M and P groups (see Figure 2). Significant 'RD by Interval (linear & quadratic)' effects (both *ps* = .022) and a marginal RD effect (*p* = .082) suggest that not only did RD+ individuals generally report higher NA than RD- individuals, but RD+ individuals also demonstrated greater increases of NA in response to the stressor tasks. Furthermore, a significant 'RD by Interval by Group' effect (*p* = .010) showed that Group modified the relationship between RD and Interval such that the M group reported less of an increase in NA in response to stress induction than P group. Figure 2 distinguishes the trajectories of NA by Group and RD status. The P group was divided into two subgroups—P/RD- and P/RD+ —and the M group was divided into two subgroups—M/RD- and M/RD+. To summarize, P/RD+ reacted to the stressor tasks with the greatest increases in NA, followed by M/RD+, then P/RD-, and lastly M/RD- was the least reactive to stress induction.

Separate analyses were conducted for each group to obtain slope coefficients for each group and to aid in the interpretation of group differences between M and P groups (see Table 11). There were no significant effects for RD or 'RD by Interval' interactions for the P and E groups. Within the M group, RD+ individuals reported greater increases in NA than RD- individuals in NA at time 2 and time 3 (*ps* < .05, see Table 11). This finding suggested that the effects of RD in the comparison between M and P groups were largely driven by RD's effects on NA in the M group.

All four fit statistics demonstrated no improvement in model goodness-of-fit with the inclusion of RD as a predictor compared to a model without RD. Given that Group effects were largely comparable between the models with and without RD, RD was not included in models assessing for testing effects.

Testing effects on NA. Testing effects were assessed by including Pre/Post as an additional predictor for NA. Pre/Post is a categorical variable that indicates whether the participant received a pre-intervention lab assessment. All group comparisons rendered significant effects found in previous analyses nonsignificant with the inclusion of Pre/Post as a predictor (see Tables 12 & 13 for group comparisons and Table 14 for beta coefficients). This finding suggested that testing effects may be collinear with the Group and Interval effects found in prior analyses.

To clarify these findings, analyses on NA were conducted excluding participants who received a Pre-intervention lab session. Consistent with initial analyses omitting Pre/Post as a predictor, group comparisons demonstrated significant linear and quadratic Interval effects for all group comparisons (*ps* all < .001, see Table 15 & 16 for group comparisons, Table 17 for beta coefficients). All groups began at comparable levels of NA and NA increased similarly at Interval 2 (first stressor). After Interval 2, the groups appeared to diverge at Interval 3 (second stressor), with NA continuing to increase for the P group and NA increasing slightly for the E group, whereas NA decreased for the M group. All groups returned to similar levels of NA at Interval 4. Significant differences in NA between M and P groups were evident across lab interval (see Figure 3). The P group appeared to show greater NA and the M group appeared to show lessened NA at Interval 3 compared to the E group, but these differences were not statistically significant. Though it appears that the E group served as a baseline measure of NA over the course of stress induction, M and P groups did not differ significantly from the E group. This pattern of NA suggested that while all groups ultimately returned to baseline levels of NA, the M group appeared to return to baseline levels of NA more quickly than the other groups.

Group Effects on Positive Affect

As with NA, two sets of analyses were required to test all possible group comparisons (analysis 1: P vs. E, M vs. E; analysis 2: P vs. M). Significant positive linear and negative

quadratic trends were evidenced for both analyses (all *p*s < .001; see Table 18, 19; for means & standard deviations see Table 2). Similar to the trajectories of NA across Interval, PA increased from baseline to stressor 1 and continued to increase from stressor 1 to stressor 2, then decreased from stressor 2 to relaxation. Figure 3 represents the changes in PA across the 4 Lab Intervals, by Group. The negative quadratic Interval effect is contrary to the hypothesis that PA would decrease in response to stress induction (Hypothesis 1b).

Analyses of contrasts between groups allowed for comparisons of treatment group. M differed significantly from both P and E groups (*ps* < .05, see Table 18 & 19). P and E groups did not differ in PA. Significant 'Group by Interval (both linear & quadratic)' effects showed that the M group displayed slower linear increases in PA across Interval (M<E, t = -2.00, p = .046; M<P, t = -2.37, p = .018), and a shallower quadratic Interval effect for PA (M>E, t = 1.88, p = .061; M>P, t = 2.56, p = .011). Separate analyses were conducted for each group to obtain slope coefficients for each group so that they could be compared (see Table 5).

M group reported relatively high levels of PA that were stable across the lab procedure. PA decreased from Interval 3 (stressor 2) to Interval 4 (recovery) for each group, however PA decreased to a lesser extent in the M group compared to the other groups.

PA, Covarying negative affect. NA was included as a covariate in the model to assess for PA independent of NA. As before, analyses were conducted to compare groups. Significant linear and quadratic Interval effects (*ps* < .01) were found only in the comparison between the M and E groups (see Tables 20 & 21 for contrasts and Table 8 for betas). The results of the analyses between M and E were largely identical to analyses of PA omitting NA as a covariate. Significant Group and 'Group x Interval (quadratic)' effects were retained (*p*s < .05), though linear and quadratic Interval effects were rendered nonsignificant and the previously significant 'Group by linear Interval' effect became marginally significant (p = .054).

All four fit statistics indicated no change in goodness of fit with the inclusion of NA as a covariate. Because covarying NA did not improve goodness of fit, NA was not included in subsequent analyses of PA.

History of Recurrent Depression on PA. In order to assess for the effects of history of recurrent depression on PA, RD was included in analyses and allowed to interact with all other predictors. Results of the group comparisons were largely comparable to analyses without RD (see Tables 22 & 23 for contrasts, Table 11 for betas). No RD or 'RD by Interval' effects were significant. RD did not interact with other predictor terms, suggesting no evidence for effects on PA due to history of recurrent depression.

Testing Effects on PA. Testing effects were evaluated by including Pre/Post as an additional predictor for PA. The significant group differences between M versus P, and M versus E groups were not preserved with the inclusion of Pre/Post as a predictor (see Table 25). The results of these contrasts were largely different than previous analyses with PA as an outcome. There was a significant main effect for Pre/Post and significant interactions with Pre/Post (see Table 24) suggesting that testing effects were present.

To clarify these findings, analyses on PA were conducted excluding participants who received a Pre-intervention lab session. Group comparisons demonstrated significant linear and quadratic Interval effects for all groups (*ps* all < .0001, see Tables 26 & 27). Significant differences in PA between M and P groups were evident across lab interval (see Figure 5). These differences were comparable to the results obtained from initial analyses on PA in which the entire sample was utilized—including participants who received pre-intervention lab assessment—and the Pre/Post variable was omitted. Additionally, these prior analyses demonstrated group differences between M and E groups, but this comparison was not significant in analyses of the post-only subset of participants.

All three groups appeared to have relatively similar levels of PA across interval 1, 2, and 3 (see Figure 7). Levels of PA seemed to diverge from Interval 3 to Interval 4. While the M group appeared to sustain consistently high levels of PA across the lab induction procedure with a slight dip in PA at Interval 4, the P group showed a drastic drop in PA at Interval 4 (recovery). This suggests that while the stressor tasks elicited comparable levels of PA in response to the stressors, the groups differed to the extent they retained those levels of PA at the final Interval (recovery).

Chapter 4

DISCUSSION

The main purpose of this study was to examine the stress-reducing effects of two active interventions for RA patients. Differences consistently emerged between the M group and P group, as well as between the M group and E attention control. Compelling evidence was found for changes in affective responding under conditions of laboratory-induced stress for RA patients who had underwent a mindfulness emotion regulation intervention, particularly when compared to RA patients who participated in the CBT-pain management intervention. Two affective outcome variables were examined—positive affect and negative affect—and significant group differences emerged for both. Overall, the M group consistently differed from the P group in many analyses. The significant differences between group M and group P persisted even after partialing out the effects of other predictors (e.g. affective covariates, history of recurrent depression). Testing effects were evident in the analyses, but the same pattern of group differences were found after excluding patients who underwent a pre-intervention lab visit. The M group differed from the E group for both NA and PA in analyses that included the entire sample, but this comparison was nonsignificant in analyses that excluded participants who received a pre-intervention laboratory assessment.

As predicted, the M group differed significantly from the P and E groups in negative affect, though not in the way anticipated. The way in which the groups differed was not characterized by the swiftness of recovery from stressors as was predicted in Hypothesis 2, but from differences in *reactivity* to stress. The trajectories of NA in the M group were characterized as consistent and non-reactive during the stress induction procedure, whereas the P group demonstrated more reactive affective responses to stress, suggesting a dampening of NA reactivity for the M group relative to the other groups. Because half of the sample received a pre-intervention laboratory session as a part of the Solomon-6 study design, testing effects were considered as a potential influence on reactivity to stress. Analysis of the post-only subset of participants confirmed that the M group exhibited less NA reactivity to stress than the P group, but not the E group. Furthermore, the M group appeared to recover more quickly from the initial

stressor than the P group, demonstrating decreases in NA and continued recovery even as the group underwent the second stress induction procedure. These results were consistent with Hypothesis 2a.

The group differences in PA were largely similar to those for NA. However, the trajectory of PA was in the opposite direction that was predicted. PA was *elicited* during the stressor tasks. It was predicted that PA would decrease in response to stress (Hypothesis 1b), but the opposite occurred—PA increased immediately following the stressor tasks. These findings indicated that the stressor tasks elicited increases in PA, suggesting all participants—regardless of group—recruited PA in response to stress. This somewhat counterintuitive reaction to negative stimuli may represent an emotional immune response (Wilson & Gilbert, 2005). Other researchers have posited that when faced with stimuli that induce negative emotional states, an individual will begin to cope immediately (DeWall & Baumeister, 2007). For example, DeWall and Baumeister (2007) found that reminders of mortality increased the accessibility of positive emotional information. The current study's findings that PA immediately increased after stress induction provide further evidence that the coping process is immediate and automatic. Increases in PA in response to stress were common to all groups, but the magnitude of these increases differed by intervention group. As with NA, the M group appeared to demonstrate less reactivity to the stressors compared to the P and E groups. This lack of PA reactivity taken together with dampened NA reactivity suggests that because the stressors did not induce negative emotions to the same degree as the other groups, the M group had less of a need to cope with the stressors and therefore did not demonstrate the same degree of increases in PA as the other groups. All groups showed similar levels of PA during the stressor tasks, but PA decreased to a lesser degree at recovery for the M group than the P and E groups. The M group's response to stress seemed to be characterized by lessened loss of PA (and less reactivity to stress) and sustained levels of PA across the lab interval. This finding is compatible with the hypothesis that the M group would fare better than the other groups (Hypothesis 2b), though it appeared that the M group demonstrated consistent sustainability of PA, rather than affective recovery from stress induction.

Compared to the other groups, the M group appeared to more capably manage negative affect and to retain positive emotions independent of the negative and potentially distressing stress induction procedure. The stress-reducing effects of mindfulness practice have been well documented (e.g., Baer, 2003) and the M group's dampened affective response to stress is consistent with recent research that shows mindfulness facilitates stress processing via lower emotional reactivity to stressors and quicker recovery from unpleasant emotional states (Weinstein, Brown, & Ryan, 2009). Several mechanisms have been proposed to explain the stress-reducing effects of mindfulness. Research with nonclinical samples suggests that the ability to manage negative emotions and non-attachment (e.g., extent that happiness and positive emotions are independent of specific outcomes and events) mediate the relationship between mindfulness and mental health (Coffey et al., 2010). Improved attention and greater parasympathetic activity have been associated with mindfulness in a randomized control trial comparing a brief 5-day mind-body integrative intervention to a relaxation control (Tang et al., 2007; Tang et al., 2009). Clarity of one's affective experience is another mechanism. Individuals high in trait mindfulness demonstrated greater widespread prefrontal cortical activation and reduced bilateral amygdala activation during affect labeling (Cresswell, Way, Eisenberger, & Lieberman, 2007). Garland, Gaylord, and Fredrickson (2011) propose that cognitive variables, such as positive reappraisal, mediate the relationship between dispositional mindfulness and reduction of stress. These are potential mediators that may help to explain the connection between mindfulness and psychological flourishing.

It is interesting that most of the differences were found between the two active interventions, rather than between the active interventions and attention control. There were no significant differences between the P and E group, suggesting that the P intervention did not contribute to any changes in affect. Indeed, the P group consistently seemed to fare worse than both groups, evidencing the greatest affective reactivity to stress—even more reactive the E group—and greatest loss of PA at recovery. Though the differences between the P and E groups were not significant, this "null result" prompts further questions about the nature of this finding. Two potential explanations emerge: first, it is possible that utilizing a cognitive approach called

even more attention to the negative aspects of experience without directly changing those aspects, thus increasing unpleasant feelings to a greater extent than if no action was taken at all. Cognitive strategies emphasized taking an evaluative approach that included closely examining maladaptive thoughts and beliefs and actively replacing them with more adaptive alternatives. This process may have been effortful and fatiguing, which could have perpetuated negative feelings even further. Perhaps cognitive strategies simply took longer to work and recovery occurred later than it would naturally without intervention. An alternative explanation is that cognitive strategies did not alter affect at all, and that pre-to-post intervention decreases in self-report pain, depressive symptoms, and pain control in this group (Zautra et al., 2008) were largely due to changes in cognition.

The findings from the present study underscore the differences between mindfulness-based and CBT-based interventions and highlight the potential value of incorporating elements of mindfulness into the gold standard CBT interventions. CBT has been shown to increase positive cognitive coping (e.g., active coping) and appraisal and reduce behavioral expressions of pain (Morley, Eccleston, & Williams, 1999), but in utilizing CBT without paying attention to emotion regulation, we may be introducing awareness of negative psychological states and affect without giving individuals the tools with which to deal with them. The addition of mindfulness techniques may supplement the benefits of cognitive behavioral therapies.

Positive affect may serve as a source of resilient responding. In a sample of women with osteoarthritis or fibromyalgia or both, patients with greater positive affect demonstrated lower negative affect during times of increased pain and stress (Zautra, Johnson, &Davis, 2005). Positive emotion has been studied as a potential source of resilience that works by undoing some of the deleterious physiological effects of negative emotion and by promoting faster recovery to baseline levels of affect (Fredrickson & Levenson, 1998). Beyond returning to homeostasis, positive affect has been shown to counteract and reverse the negative effects of what Tice and colleagues (2007) term “ego-depletion.” They posit that self-regulation is integral for functioning, but requires the use of psychological resources. Depletion of these stores is counteracted by positive emotion, which replenishes and expands the capacity to self-regulate. Utilizing positive

affect may be a natural coping response that those who are well-regulated (e.g., individuals well versed in mindfulness practice) are especially able to capitalize on. Emphasizing the cultivation of positive emotion, whether it be by attending to and savoring the positive aspects of experience from the mindfulness perspective, or by engaging in pleasant events scheduling as prescribed by the cognitive behavioral tradition, is integral for the promotion of emotion regulation.

Attention needs to be paid to the effects of depression history, as individuals with a history of depression by definition showed deficits in emotion regulation. Hypothesis 3 examined the effects of a history of recurrent depression. Interestingly, RD only affected the trajectory of NA, not PA. This lends credence to the assertion that NA and PA are independent, albeit related constructs (Reich et al., 2003). Furthermore, history of recurrent depression only affected participants in the M group. Participants in the M group without a history of recurrent depression were less reactive to the stressor tasks than those with a history of depression. This supports the preposition that depression history may serve as a risk factor in terms of susceptibility to stress. This finding, coupled with the previous finding that M/RD+ group demonstrated pre to post changes in increases in PA, reductions in NA, greater coping efficacy, and lessened catastrophizing in diary data, suggests that the emotion regulation benefits of mindfulness-based techniques (Zautra et al., 2008) may be especially helpful for RD+ individuals, by expanding their repertoire of emotion regulation strategies. Positive emotions have been found to mediate the relationship between dispositional mindfulness and depressive symptoms in nonclinical individuals (Jimenez, Niles, & Park, 2010).

There are several limitations in this study. One challenge faced was disentangling testing effects that were a result of the Solomon-6 study design. This study would have been strengthened by including pre-intervention laboratory stress induction data, so that pre-post effects could be examined. However, the presence of testing effects may very well diminish pre-post effects. There is some evidence to suggest that the testing effects found in this study were collinear with group effects; however, more work to clarify this assertion is needed. Another limitation of the current study is that recovery from stress was difficult to assess in the M group, because they were not as emotionally reactive as the other groups. Although this result was

valuable in and of itself, the muted reaction to stress induction made it difficult to examine recovery processes. Induction of positive, negative, and neutral emotional states may be useful in future research. Also, given that RA patients have chronic pain, examination of pain induction procedures and their effects on stress and affect would be especially relevant. While controlled laboratory paradigms offer strengths in standardization, they are often criticized for their lack of ecological validity, which is why a multi-pronged approach in which data is collected in multiple forms (e.g., diary data) and time frames is valuable. The findings from the current, laboratory-based study taken together with the results of the daily diary analyses from the parent study (see Zautra et al., 2008) offer a nuanced and complete picture of how mindfulness and cognitive-behavioral approaches affect emotion regulation in RA patients.

Many advances have been made in research on mindfulness, yet many unanswered questions remain about the mechanisms involved in the processes underlying mindfulness. This study adds to understanding of how mindfulness promotes resilient functioning in chronic pain patients. We have already seen that RA patients who underwent the mindfulness in the study reported pre to post intervention changes in a variety of outcomes. The current investigation further clarified the regulatory functions of mindfulness, while simultaneously inviting further inquiry about processes of recovery and sustainability of affect.

REFERENCES

Astin, J. A., Beckner, W., Soeken, K., Hochberg, M. C., & Berman, B. (2002). Psychological interventions for rheumatoid arthritis: A meta-analysis of randomized controlled trials *Arthritis and Rheumatism, 47*(3), 291-302.

Baer, R. A. (2003). Mindfulness training as a clinical intervention: A conceptual and empirical review. *Clinical Psychology: Science and Practice, 10*(2), 125-143.

Brown, K. W., Ryan, R. M., & Creswell, J. D. (2007). Mindfulness: Theoretical foundations and evidence for its salutary effects. *Psychological Inquiry, 18*(4), 211-237.

Carmody, J., & Baer, R. A. (2008). Relationships between mindfulness practice and levels of mindfulness, medical and psychological symptoms and well-being in a mindfulness-based stress reduction program. *Journal of Behavioral Medicine, 31*(1), 23-33.

Chambers, R., Lo, B. C. Y., & Allen, N. B. (2008). The impact of intensive mindfulness training on attentional control, cognitive style, and affect. *Cognitive Therapy and Research, 32*(3), 303-322.

Chida, Y., & Hamer, M. (2008). Chronic psychosocial factors and acute physiological responses to laboratory-induced stress in healthy populations: A quantitative review of 30 years of investigations. *Psychological Bulletin, 134*(6), 829-885.

Coffey, K.A., Hartman, M., & Fredrickson, B.L. (2010). Deconstructing mindfulness and constructing mental health: Understanding mindfulness and its mechanism of action. *Mindfulness, 1*, 235-253.

Conner, T. S., Tennen, H., Zautra, A. J., Affleck, G., Armeli, S., & Fifield, J. (2006). Coping with rheumatoid arthritis pain in daily life: Within-person analyses reveal hidden vulnerability for the formerly depressed. *Pain, 126*(1-3), 198-209.

Cresswell, J. D., Way, B. M., Eisenberger, N. I., & Lieberman, M. D. (2007). Neural correlates of dispositional mindfulness during affect labeling. *Psychosomatic Medicine, 69*, 560-565.

Davis, M. C. (1999). Oral contraceptive use and hemodynamic, lipid, and fibrinogen responses to smoking and stress in women. *Health Psychology, 18*(2), 122-130.

Davis, M. C., Twamley, E. W., Hamilton, N. A., & Swan, P. D. (1999). Body fat distribution and hemodynamic stress responses in premenopausal obese women: A preliminary study. *Health Psychology, 18*(6), 625-633.

Davis, M. C., Zautra, A. J., & Reich, J. W. (2001). Vulnerability to stress among women in chronic pain from fibromyalgia and osteoarthritis. *Annals of Behavioral Medicine, 23*(3), 215-226.

Davis, M. C., Zautra, A. J., & Smith, B. W. (2004). Chronic pain, stress, and the dynamics of affective differentiation. *Journal of Personality.Special Issue: Emotions, Personality, and Health, 72*(6), 1133-1159.

Davis, M. C., Zautra, A. J., Younger, J., Motivala, S. J., Attrep, J., & Irwin, M. R. (2008). Chronic stress and regulation of cellular markers of inflammation in rheumatoid arthritis: Implications for fatigue. *Brain, Behavior, and Immunity, 22*(1), 24-32.

DeWall, C. N., & Baumeister, R. F. (2007). From terror to joy: Automatic tuning to positive affective information following mortality salience. *Psychological Science, 18*(11), 984-990.

Dickens, C., McGowan, L., Clark-Carter, D., & Creed, F. (2002). Depression in rheumatoid arthritis: A systematic review of the literature with meta-analysis. *Psychosomatic Medicine, 64*(1), 52-60.

Feldmann, M., & Maini, R. N. (2008). Role of cytokines in rheumatoid arthritis: An education in pathophysiology and therapeutics. *Immunological Reviews, 223*(1), 7-19.

Fredrickson, B. L., & Levenson, R. W. (1998). Positive emotions speed recovery from the cardiovascular sequelae of negative emotions. *Cognition and Emotion, 12*, 191-220.

Garland, E. L., Gaylord, S. A., & Fredrickson, B. L. (2011). Positive reappraisal mediates the stress-reductive effects of mindfulness: An upward spiral process. *Mindfulness, 2*,59-67.

Herrmann, M., Scholmerich, J., & Straub, R. (2000). Stress and rheumatic diseases. *Rheumatic Disease Clinics of North America, 26*(4), 737-763.

Kabat-Zinn, J. (1982). An outpatient program in behavioral medicine from chronic pain patients based on the practice of mindfulness medication: Theoretical considerations and preliminary results. *General Hospital Psychiatry, 4*(1), 33-47.

Jimenez, S. S., Niles, B. L., & Park, C. L. (2010). A mindfulness model of affect regulation and depressive symptoms: Positive emotions, mood regulation expectancies, and self-acceptance as regulatory mechanisms. *Personality and Individual Differences, 49*, 645-650.

Land, H., Verdurmen, J., Have, M., Dorsselaer, S., Beekman, A., & Graaf, R. (2010). The association between arthritis and psychiatric disorders; results from a longitudinal population-based study *Journal of Psychosomatic Research, 68*(2), 187-193.

Littell, R. C., Milliken, G. A., Strong, W. W., & Wolfinger, R. D. (1996). *SAS system for mixedmodels*. Cary, NC: SAS Institute, Inc.

McCracken, L. M., Gauntlett-Gilbert, J., & Vowles, K. E. (2007). The role of mindfulness in a contextual cognitive-behavioral analysis of chronic pain-related suffering and disability. *Pain, 131*(1-2), 63-69.

Morley, S., Eccleston, C., & Williams, A. (1999). Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain, 80,* 1-13.

National Institutes of Health (2009). Handout on health: rheumatoid arthritis. From http://www.niams.nih.gov/Health_Info/Rheumatic_Disease/.

Nicassio, P. M. (2010). Arthritis and psychiatric disorders: Disentangling the relationship. *Journal of Psychosomatic Research, 68*(2), 183-185.

Olmsted, M. E., Finan, P. H., Zautra, A. J., Davis, M. C., & Parrish, B. P. (unpublished). Testing effects in psychological challenge: A case for using a solomon-four group design.

Reich, J.W., Zautra, A. J., & Davis, M.C., (2003). Dimensions of affect relationships: Models and their integrative implications. *Review of General Psychology, 7*(1), 66-83. doi:10.1037/10899-2680.7.1.66

Rohde, P., Lewinsohn, P. M., & Seeley, J. R. (1997). Comparability of telephone and face-to-face interviews in assessing axis I and II disorders. *American Journal of Psychiatry, 154*(11), 1593-1598.

Rosenzweig, S., Greeson, J. M., Reibel, D. K., Green, J. S., Jasser, S. A., & Beasley, D. (2010). Mindfulness-based stress reduction for chronic pain conditions: Variation in treatment outcomes and role of home meditation practice. *Journal of Psychosomatic Research, 68*(1), 29-36.

Shapiro, S. L., Oman, D., Thoresen, C. E., Plante, T. G., & Flinders, T. (2008). Cultivating mindfulness: Effects on well-being. *Journal of Clinical Psychology, 64*(7), 840-862.

Simon, G. E., Revicki, D., & VonKorff, M. (1993). Telephone assessment of depression severity. *Journal of Psychiatric Research, 27*(3), 247-252.

Teasdale, J. D., Moore, R. G., Hayhurst, H., Pope, M., Williams, S., & Segal, Z. V. (2002). Metacognitive awareness and prevention of relapse in depression: Empirical evidence. *Journal of Consulting and Clinical Psychology, 70*(2), 275-287.

Teasdale, J. D., Segal, Z. V., Williams, J. M. G., Ridgeway, V. A., Soulsby, J. M., & Lau, M. A. (2000). Prevention of relapse/recurrence in major depression by mindfulness-based cognitive therapy. *Journal of Consulting and Clinical Psychology, 68*(4), 615-623.

Tice, D. M., Baumeister, R. F., Shmueli, D., & Muraven, M. (2007). Restoring the self: Positive affect helps improve self-regulation following ego depletion. *Journal of Experimental Social Psychology, 43*, 379-384.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and validation of brief measures of positive and negative affect: The PANAS scales. *Journal of Personality and Social Psychology, 54*(6), 1063-1070.

Weinstein, N., Brown, K. W., & Ryan, R. M. (2009). A multi-method examination of the effects of mindfulness on attribution, coping, and emotional well-being. *Journal of Research in Personality, 43*, 374-385.

Wilson, T. D., & Gilbert, D. T. (2005). Affective forecasting: Knowing what to want. *Current Directions in Psychological Science, 14*(3), 131-134.

Zautra, A. J., Yocum, D. C., Villanueva, I., Smith, B., Davis, M. C., Attrep, J., et al. (2004). Immune activation and depression in women with rheumatoid arthritis *The Journal of Rheumatology, 31*(3), 457-463.

Zautra, A. J., Affleck, G. G., Tennen, H., Reich, J. W., & Davis, M. C. (2005). Dynamic approaches to emotions and stress in everyday life: Bolger and zuckerman reloaded with positive as well as negative affects. *Journal of Personality.Special Issue: Advances in Personality and Daily Experience, 73*(6), 1511-1538.

Zautra, A. J., Burleson, M. H., Smith, C. A., Blalock, S. J., Wallston, K. A., DeVellis, R. F., et al. (1995). Arthritis and perceptions of quality of life: An examination of positive and negative affect in rheumatoid arthritis patients. *Health Psychology, 14*(5), 399-408.

Zautra, A. J., Davis, M. C., Reich, J. W., Nicassario, P., Tennen, H., Finan, P., et al. (2008). Comparison of cognitive behavioral and mindfulness meditation interventions on adaptation to rheumatoid arthritis for patients with and without history of recurrent depression. *Journal of Consulting and Clinical Psychology, 76*(3), 408-421.

Zautra, A. J., Hamilton, N. A., Potter, P., & Smith, B. (1999). Field research on the relationship between stress and disease activity in rheumatoid arthritis. *Annals of the New York Academy of Sciences, 876*, 397-412.

Zautra, A. J., Hoffman, J., Potter, P., Matt, K. S., Yocum, D., & Castro, L. (1997). Examination of changes in interpersonal stress as a factor in disease exacerbations among women with rheumatoid arthritis. *Annals of Behavioral Medicine, 19*(3), 279-286.

Zautra, A.J., Johnson, L.M., & Davis, M.C. (2005). Positive affect as a source of resilience for women in chronic pain. *Journal of Consulting and Clinical Psychology, 73*(2), 212-230.

Zautra, A. J., Parrish, B. P., Van Puymbroeck, C. M., Tennen, H., Davis, M. C., Reich, J. W., et al. (2007). Depression history, stress, and pain in rheumatoid arthritis patients. *Journal of Behavioral Medicine, 30*(3), 187-197.

Zautra, A., Smith, B., Affleck, G., & Tennen, H. (2001). Examinations of chronic pain and affect relationships: Applications of a dynamic model of affect. *Journal of Consulting and Clinical Psychology*, *69*(5), 786-795.

Table 1

Demographic Characteristics By Group

Characteristic	Group		
	M (*n* = 39)	P (*n* = 48)	E (*n* = 41)
Gender, *n*			
Male	17	15	8
Female	22	33	33
Ethnicity, *n*			
White	34	41	33
Other	4	7	6
Age in years, *M* (*SD*)	57.13 (15.16)	54.65 (12.68)	52.02 (12.43)
Years with RA diagnosis, *M* (*SD*)	11.34 (11.07)	15.43 (13.91)	11.55 (13.87)
Median family income	$40,000 - $49,999	$40,000 - $49,999	$50,000 - $59,999
History of recurrent depression, *n*			
0 or 1 depressive episode	31	32	27
2 or more depressive episodes	7	16	14

Table 2

Means (Standard Deviations) for NA and PA Across Lab Interval by Group

Outcome measure	Group		
	M	P	E
Negative affect			
Lab interval 1	1.15 (.31)	1.20 (.52)	1.15 (.30)
Lab interval 2	1.49 (.65)	1.74 (.69)	1.83 (.72)
Lab interval 3	1.63 (.63)	2.06 (.88)	1.92 (.77)
Lab interval 4	1.07 (.16)	1.16 (.35)	1.20 (.37)
Positive affect			
Lab interval 1	2.82 (.73)	2.45 (.88)	2.49 (.88)
Lab interval 2	2.97 (.70)	2.91 (.87)	2.80 (.75)
Lab interval 3	3.10 (.76)	2.89 (.83)	3.11 (.78)
Lab interval 4	2.58 (.87)	2.13 (.92)	2.36 (.93)

Table 3

Group and interval effects in the prediction of NA, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.07	.01	5.33	<.001***
Var (1)		.09	.02	6.28	<.001***
Var (2)		.43	.06	7.41	<.001***
Var (3)		.53	.07	7.74	<.001***
Var (4)		.03	.01	2.81	.003**
ARH (1)		.20	.07	2.81	.005**
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		-.25	.19	-1.34	.18
Between subject (*df* = 125)					
Group	P vs. E	.08	.26	0.31	.76
	M vs. E	.55	.27	2.01	.047*
Within subject (*df* = 376)					
Interval		1.75	.21	8.37	<.001**
Interval*Interval		-.35	.04	-8.41	<.001**
Within & between subject interaction (*df* = 376)					
Group*Interval	P vs. E	-.03	.28	-0.09	.93
	M vs. E	-.67	.30	-2.24	.03*
Group*Interval*Interval	P vs. E	-.002	.06	-0.03	.98
	M vs. E	.13	.06	2.12	.03*

†*p*<.10 **p*<.05 ***p*<.01 ****p*<.001

Table 4

Group and interval effects in the prediction of NA, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.10	.03	4.01	<.001***
Residual		.24	.02	11.31	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		.23	.22	1.03	.30
Between subject (*df* = 85)					
Group	P vs. M	-.53	.30	-1.78	.08†
Within subject (*df* = 253)					
Interval		1.12	.20	5.64	<.001***
Interval*Interval		-.23	.04	-5.77	<.001***
Within & between subject interaction (*df* = 253)					
Group*Interval	P vs. M	.69	.27	2.59	.01*
Group*Interval*Interval	P vs. M	-.13	.05	-2.53	.01*

†p<.10 *p<.05 **p<.01 ***p<.001

Table 5

Beta Coefficients (Standard Error) for Linear & Quadratic Interval Effects on NA and PA by Group

Fixed effects	M[a]		P[b]		E[c]	
	ß (SE)	*t*	ß (SE)	*t*	ß (SE)	*t*
Negative affect						
Interval	1.11 (.19)	5.88	1.81 (.20)	9.11	1.77 (.19)	9.45
Interval*Interval	-.23 (.04)	-6.03	-.36 (.04)	-9.16	-.35 (.04)	-9.46
Positive affect						
Interval	.76 (.18)	4.27	1.42 (.20)	7.25	1.37 (.23)	6.03
Interval*Interval	-.17 (.04)	-4.66	-.30 (.04)	-7.88	-.28 (.04)	-6.18

Note: [a] $df_M = 114$. [b] $df_P = 142$. [c] $df_E = 120$. All *p*-values < .001

Table 6

Group and interval effects in the prediction of NA covarying PA, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.07	.01	5.22	<.001***
Var (1)		.10	.02	6.26	<.001***
Var (2)		.44	.06	7.29	<.001***
Var (3)		.51	.07	7.64	<.001***
Var (4)		.03	.01	2.84	.002**
ARH (1)		.21	.07	2.95	.003**
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		-.39	.42	-0.93	.35
Between subject (*df* = 125)					
Group	P vs. E	.07	.26	0.28	.78
	M vs. E	.48	.27	1.78	.08†
Within subject (*df* = 373)					
PA		.07	.13	0.55	.58
Interval		1.77	.47	3.77	<.001***
Interval*Interval		-.35	.09	-3.78	<.001***
Within & between subject interaction (*df* = 373)					
PA x Interval		-.03	.15	-0.22	.83
PA*Interval*Interval		.007	.03	0.24	.81
Group*Interval	P vs. E	-.01	.28	-0.05	.96
	M vs. E	-.62	.30	-2.06	.04*
Group*Interval*Interval	P vs. E	-.003	.06	-0.06	.95

M vs. E	.12	.06	1.95	.05†

†$p<.10$ *$p<.05$ **$p<.01$ ***$p<.001$

Table 7

Group and interval effects in the prediction of NA covarying PA, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.10	.03	4.02	<.001***
Residual		.24	.02	11.25	<.001***
Fixed effects					
Predictor variable	Contrast	β	*SE*	*t*	*p*
Intercept		.34	.48	0.59	.56
Between subject (*df* = 85)					
Group	P vs. M	-.49	.31	-1.61	.11
Within subject (*df* = 253)					
PA		-.02	.19	-0.08	.93
Interval		.88	.53	1.65	.10
Interval*Interval		-.19	.10	-1.85	.07†
Within & between subject interaction (*df* = 253)					
PA*Interval		.06	.17	0.35	.73
PA*Interval*Interval		-.006	.03	-0.19	.84
Group*Interval	P vs. M	.66	.27	2.42	.02*
Group*Interval*Interval	P vs. M	-.12	.05	-2.30	.02*

†*p*<.10 **p*<.05 ***p*<.01 ****p*<.001

Table 8

Beta Coefficients (Standard Error) for PA (Covarying NA), and NA (Covarying PA) by Group

Fixed effects	M[a]			P[b]			E[c]		
	ß (SE)	*t*	*p*	ß (SE)	*t*	*p*	ß (SE)	*t*	*p*
Negative affect									
Interval	1.46 (.70)	2.09	.04*	1.21 (.67)	1.81	.07†	2.44 (.70)	3.47	<.001***
Interval*Interval	-.29 (.13)	-2.17	.03*	-.26 (.13)	-1.98	.0496*	-.50 (.14)	-3.62	<.001***
PA	.16 (.26)	.62	.54	-.12 (.26)	-.45	.65	.20 (.26)	.74	.46
PA*Interval	-.13 (.23)	-.54	.59	.16 (.24)	.69	.49	-.25 (.25)	-1.01	.31
PA*Interval*Interval	.03 (.05)	.57	.57	-.02 (.05)	-.51	.61	.06 (.05)	1.16	.25
Positive affect									
Interval	.88 (.78)	1.13	.26	.79 (.61)	1.29	.20	1.96 (.77)	2.55	.01*
Interval*Interval	-.25 (.16)	-1.54	.13	-.19 (.13)	-1.47	.14	-.45 (.15)	-3.02	.003**
NA	.11 (.77)	.14	.89	-.18 (.48)	-.39	.70	.23 (.73)	.32	.75
NA*Interval	-.29 (.67)	-.43	.67	.27 (.43)	-.63	.53	-.47 (.58)	-.81	.42
NA*Interval*Interval	.11 (.14)	.79	.43	-.05 (.09)	-.54	.59	.14 (.11)	1.26	.21

Note: [a] $df_M = 111$, [b] $df_P = 139$, [c] $df_E = 117$, †$p<.10$ *$p<.05$ **$p<.01$ ***$p<.001$

Table 9

Group, interval, and RD effects in the prediction of NA, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.06	.01	4.95	<.001***
Var (1)		.08	.01	6.08	<.001***
Var (2)		.44	.06	7.21	<.001***
Var (3)		.50	.07	7.53	<.001***
Var (4)		.03	.01	3.48	<.001***
ARH (1)		.18	.07	2.56	.01*
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		-.21	.23	-0.90	.37
Between subject (*df* = 121)					
RD		-.14	.39	-0.37	.71
Group	P vs. E	-.12	.31	-0.37	.71
	M vs. E	.65	.31	2.09	.04*
Within subject (*df* = 367)					
Interval		1.62	.26	6.33	<.001***
Interval*Interval		-.32	.05	-6.28	<.001***
Within & between subject interaction (*df* = 367)					
RD*Group	P vs. E	.54	.53	1.01	.32
	M vs. E	-.82	.63	-1.29	.20
RD*Interval		.39	.44	0.90	.37
RD*Interval*Interval		-.09	.09	-1.01	.31
Group*Interval	P vs. E	.08	.35	0.22	.83

	M vs. E	-.75	.35	-2.13	.03*
Group*Interval*Interval	P vs. E	-.02	.07	-0.29	.78
	M vs. E	.14	.07	1.99	.047*
RD*Group*Interval	P vs. E	-.24	.59	-0.40	.69
	M vs. E	.77	.70	1.10	.27
RD*Group*Interval *Interval	P vs. E	.04	.12	0.34	.73
	M vs. E	-.14	.14	-1.03	.30

†$p<.10$ *$p<.05$ **$p<.01$ ***$p<.001$

Table 10

Group, interval, and RD effects in the prediction of NA, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.08	.02	3.50	<.001***
Residual		.24	.02	11.16	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		.41	.25	1.63	.11
Between subject (*df* = 82)					
RD		-1.02	.58	1.76	.08†
Group	P vs. M	-.80	.35	-2.29	.02*
Within subject (*df* = 249)					
Interval		.90	.22	4.01	<.001***
Interval*Interval		-.18	.04	-4.13	<.001***
Within & between subject interaction (*df* = 249)					
RD*Group	P vs. M	1.29	.72	1.79	.08†
RD*Interval		1.20	.52	2.31	.02*
RD*Interval*Interval		-.24	.10	-2.30	.02*
Group*Interval	P vs. M	.84	.31	2.69	.008**
Group*Interval*Interval	P vs. M	-.16	.06	-2.59	.01*
RD*Group*Interval	P vs. M	-.97	.64	-1.51	.13
RD*Group*Interval *Interval	P vs. M	.18	.13	1.42	.16

†p<.10 *p<.05 **p<.01 ***p<.001

Table 11

Beta Coefficients (Standard Error) for Analyses Including History of Recurrent Depression (2 or more MDE)

Fixed effects	M[a]			P[b]			E[c]		
	ß (SE)	*t*	*p*	ß (SE)	*t*	*p*	ß (SE)	*t*	*p*
Negative affect									
Interval	.90 (.18)	4.88	<.001***	1.74 (.25)	7.09	<.001***	1.59 (.23)	6.88	<.001***
Interval*Interval	-.18 (.48)	-5.02	<.001***	-.34 (.05)	-7.07	<.001***	-.31 (.05)	-6.90	<.001***
RD	-1.02 (.48)	-2.13	.04*	.27 (.47)	.57	.57	-.36 (.45)	-.80	.43
RD*Interval	1.20 (.43)	2.81	.006**	.23 (.42)	.54	.59	.52 (.39)	1.32	.19
RD*Interval*Interval	-.23 (.08)	-2.79	.006**	-.05 (.08)	-.65	.51	-.10 (.08)	-1.28	.20
Positive affect									
Interval	.90 (.21)	4.38	<.001***	1.42 (.24)	5.85	<.001***	1.41 (.28)	5.03	<.001***
Interval*Interval	-.19 (.04)	-4.73	<.001***	-.30 (.05)	-6.31	<.001***	-.28 (.06)	-5.00	<.001***
RD	.31 (.59)	.52	.60	-.07 (.51)	-.13	.89	.26 (.56)	.46	.65
RD*Interval	-.49 (.48)	-1.02	.31	.02 (.42)	.06	.95	-.12 (.48)	-.25	.80
RD*Interval*Interval	.10 (.09)	1.01	.31	-.01 (.08)	-.14	.89	-.0003 (.09)	.00	.997

†p<.10 *p<.05 **p<.01 ***p<.001

Table 12

Group, interval, and testing effects in the prediction of NA, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.06	.01	5.26	<.001***
Var (1)		.09	.01	6.28	<.001***
Var (2)		.42	.06	7.30	<.001***
Var (3)		.49	.06	7.52	<.001***
Var (4)		.03	.009	3.00	.001**
ARH (1)		.16	.07	2.18	.03*
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		.37	.61	0.61	.54
Between subject (*df* = 125)					
Pre/Post		-.41	.38	-1.09	.28
Group	P vs. E	.49	.83	0.59	.56
	M vs. E	.004	.84	0.00	.996
Within subject (*df* = 367)					
Interval		.92	.66	1.38	.17
Interval*Interval		-.20	.13	-1.51	.13
Within & between subject interaction (*df* = 367)					
Pre/Post*Group	P vs. E	-.27	.51	-0.53	.60
	M vs. E	.34	.55	.62	.53
Pre/Post*Interval		.54	.41	1.33	.19
Pre/Post*Interval*Interval		-.10	.08	-1.21	.23
Group*Interval	P vs. E	-.75	.90	-0.84	.40

	M vs. E	-.32	.91	-0.35	.73
Group*Interval*Interval	P vs. E	.16	.18	0.93	.35
	M vs. E	.08	.18	0.45	.65
Pre/Post*Group*Interval	P vs. E	.48	.56	0.86	.39
	M vs. E	-.19	.60	-0.31	.76
Pre/Post*Group*Interval *Interval	P vs. E	-.11	.11	-1.00	.32
	M vs. E	.01	.12	0.17	.87

†$p<.10$ *$p<.05$ **$p<.01$ ***$p<.001$

Table 13

Group, interval, and testing effects in the prediction of NA, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.08	.02	3.70	<.001***
Residual		.23	.02	11.23	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		.15	.66	0.23	.81
Between subject (*df* = 85)					
Pre/Post		.05	.45	0.12	.90
Group	P vs. M	.83	.92	0.91	.37
Within subject (*df* = 250)					
Interval		.74	.59	1.25	.21
Interval*Interval		-.13	.12	-1.12	.26
Within & between subject interaction (*df* = 250)					
Pre/Post*Group	P vs. M	-.89	.60	0.12	.90
Pre/Post*Interval		.28	.40	0.69	.49
Pre/Post*Interval*		-.07	.08	-0.88	.38
Group*Interval	P vs. M	-.66	.82	-0.80	.42
Group*Interval*Interval	P vs. M	.11	.26	0.65	.51
Pre/Post*Group*Interval	P vs. M	.85	.54	1.58	.12
Pre/Post*Group*Interval *Interval	P vs. M	-.15	.11	-1.39	.17

†*p*<.10 **p*<.05 ***p*<.01 ****p*<.001

Table 14

Beta Coefficients (Standard Error) for Analyses Assessing for Testing Effects

Fixed effects	M[a]			P[b]			E[c]		
	ß (SE)	*t*	*p*	ß (SE)	*t*	*p*	ß (SE)	*t*	*p*
Negative affect									
Interval	.63 (.56)	1.12	.26	.08 (.63)	.12	.90	.93 (.61)	1.53	.13
Interval*Interval	-.12 (.11)	-1.10	.27	-.02 (.12)	-.20	.84	-.20 (.12)	-1.66	.0995†
Pre/Post	-.06 (.33)	-.20	.85	-.83 (.44)	-1.90	.06†	-.42 (.42)	-.99	.33
Pre/Post*Interval	.34 (.39)	.88	.38	1.13 (.39)	2.87	.005**	.55 (.37)	1.46	.15
Pre/Post*Interval*Interval	-.07 (.08)	-.96	.34	-.22 (.08)	-2.81	.006**	-.10 (.07)	-1.32	.19
Positive affect									
Interval	.10 (.55)	1.81	.07†	1.02 (.64)	1.61	.11	2.79 (.73)	3.82	<.001***
Interval*Interval	-.22 (.11)	-2.05	.04*	-.19 (.13)	-1.56	.12	-.54 (.14)	-3.72	<.001***
Pre/Post	.33 (.46)	.73	.47	.15 (.47)	.32	.75	1.38 (.53)	2.61	.01*
Pre/Post*Interval	-.16 (.38)	-.43	.66	.25 (.39)	.64	.52	-.92 (.45)	-2.04	.04*
Pre/Post*Interval*Interval	.04 (.07)	.54	.59	-.07 (.08)	-.91	.37	.17 (.09)	1.90	.06†

Note: [a] $df_M = 112$, [b] $df_P = 139$, [c] $df_E = 118$, †$p<.10$ *$p<.05$ **$p<.01$ ***$p<.001$

Table 15

Group and interval effects in the prediction of NA excluding patients who had pre lab, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.11	.03	3.69	<.001***
Var (1)		.15	.03	4.22	<.001***
Var (2)		.52	.10	4.98	<.001***
Var (3)		.61	.12	5.17	<.001***
Var (4)		.05	.02	2.28	.01*
ARH (1)		.21	.11	1.97	.049*
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		-.44	.29	-1.51	.14
Between subject (*df* = 60)					
Group	P vs. E	-.0003	.40	-0.00	.99
	M vs. E	.68	.46	1.46	.15
Within subject (*df* = 181)					
Interval		2.00	.31	6.41	<.001***
Interval*Interval		-.39	.06	-1.38	<.001***
Within & between subject interaction (*df* =181)					
Group*Interval	P vs. E	.13	.42	0.30	.76
	M vs. E	-.69	.49	-1.38	.17
Group*Interval*Interval	P vs. E	-.04	.08	-0.46	.65
	M vs. E	.12	.10	1.23	.22

†*p*<.10 **p*<.05 ***p*<.01 ****p*<.001

Table 16

Group and interval effects in the prediction of NA excluding patients who had pre lab, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.14	.05	2.69	.004**
Residual		.33	.04	7.66	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*P*
Intercept		.26	.42	0.62	.54
Between subject (*df* = 39)					
Group	P vs. M	-.94	.53	-1.77	.09†
Within subject (*df* = 117)					
Interval		1.30	.38	3.43	<.001***
Interval*Interval		-.27	.07	-3.20	<.001***
Within & between subject interaction (*df* = 117)					
Group*Interval	P vs. M	1.03	.48	2.17	.03*
Group*Interval*Interval	P vs. M	-.19	.09	-2.01	.047*

†$p<.10$ *$p<.05$ **$p<.01$ ***$p<.001$

Table 17

Beta Coefficients (Standard Error) for Post-only Analyses by Group

Fixed effects	M[a] (*n* = 15)		P[b] (*n* = 26)		E[c] (*n* = 22)	
	ß (SE)	*t*	ß (SE)	*t*	ß (SE)	*t*
Negative affect						
Interval	1.30 (.34)	3.84***	2.33 (.30)	7.67***	2.02 (.25)	8.14***
Interval*Interval	-.27 (.07)	-4.05***	-.46 (.06)	-7.69***	-.39 (.05)	8.06***
Positive affect						
Interval	.68 (.25)	2.73**	1.53 (.28)	5.38***	.94 (.24)	3.87***
Interval*Interval	-.14 (.05)	-2.91**	-.33 (.06)	-6.01***	-.20 (.05)	-4.14***

Note: [a] $df_M = 42$, [b] $df_P = 75$, [c] $df_E = 64$, *$p<.05$ **$p<.01$ ***$p<.001$

Table 18

Group and interval effects in the prediction of PA, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.43	.06	6.78	<.001***
Residual		.27	.02	13.70	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		1.35	.25	5.40	<.001***
Between subject (*df* = 125)					
Group	P vs. E	-.03	.34	-0.08	.94
	M vs. E	.84	.36	2.34	.02*
Within subject (*df* = 376)					
Interval		1.37	.21	6.61	<.001***
Interval*Interval		-.28	.04	-6.77	<.001***
Within & between subject interaction (*df* = 376)					
Group*Interval	P vs. E	.05	.28	0.18	.86
	M vs. E	-.60	.30	-2.00	.046*
Group*Interval*Interval	P vs. E	-.03	.06	-0.49	.62
	M vs. E	.11	.06	1.88	.06†

†*p*<.10 **p*<.05 ***p*<.01 ****p*<.001

Table 19

Group and interval effects in the prediction of PA, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.44	.08	5.71	<.001***
Residual		.25	.02	11.32	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		2.18	.25	8.86	<.001***
Between subject (*df* = 85)					
Group	P vs. M	-.86	.33	-2.60	.01*
Within subject (*df* = 256)					
Interval		.78	.20	3.82	<.001***
Interval*Interval		-.17	.04	-4.18	<.001***
Within & between subject interaction (*df* = 256)					
Group*Interval	P vs. M	.65	.27	2.37	.02*
Group*Interval*Interval	P vs. M	-.14	.05	-2.56	.01*

Table 20

Group and interval effects in the prediction of PA covarying NA, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.43	.06	6.80	<.001***
Residual		.27	.02	13.65	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		1.38	.48	2.86	.005**
Between subject (*df* = 125)					
Group	P vs. E	.02	.34	0.05	.96
	M vs. E	.82	.35	2.32	.02*
Within subject (*df* = 373)					
NA		.09	.34	0.26	.30
Interval		1.42	.43	3.30	.001**
Interval*Inteval		-.33	.09	-3.71	<.001***
Within & between subject interaction (*df* = 373)					
NA*Interval		-.17	.29	-0.58	.56
NA*Interval*Interval		.06	.06	1.07	.29
Group*Interval	P vs. E	.003	.28	0.01	.99
	M vs. E	-.59	.30	-1.99	.048*
Group*Interval*Interval	P vs. E	-.02	.05	-0.31	.76
	M vs. E	.11	.06	1.93	.05†

†p<.10 *p<.05 **p<.01 ***p<.001

Table 21

Group and interval effects in the prediction of PA covarying NA, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.44	.08	5.71	<.001***
Residual		.24	.02	11.25	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		2.50	.52	4.85	<.001***
Between subject (*df* = 85)					
Group	P vs. M	-.76	.33	-2.29	.02*
Within subject (*df* = 253)					
NA		-.14	.39	-0.37	.71
Interval		.43	.46	0.94	.35
Interval*Interval		-.12	.09	-1.25	.21
Within & between subject interaction (*df* = 253)					
NA*Interval		.15	.34	0.43	.67
NA*Interval*Interval		-.01	.07	-0.17	.86
Group*Interval	P vs. M	.53	.28	1.94	.05†
Group*Interval*Interval	P vs. M	-.12	.05	-2.16	.03*

†*p*<.10 **p*<.05 ***p*<.01 ****p*<.001

Table 22

Group, interval, and RD effects in the prediction of PA, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.44	.07	6.67	<.001***
Residual		.28	.02	13.54	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		1.26	.31	4.07	<.001***
Between subject (*df* = 121)					
RD		.26	.53	0.50	.62
Group	P vs. E	.09	.42	0.20	.84
	M vs. E	.86	.42	2.04	.04*
Within subject (*df* = 367)					
Interval		1.41	.26	5.48	<.001***
Interval*Interval		-.28	.05	-5.45	<.001***
Within & between subject interaction (*df* = 367)					
RD*Group	P vs. E	-.33	.72	-0.46	.65
	M vs. E	.04	.85	0.05	.96
RD*Interval		-.12	.44	-0.28	.78
RD*Interval*Interval		.0004	.09	0.00	.997
Group*Interval	P vs. E	.003	.35	0.01	.99
	M vs. E	-.51	.35	-1.45	.15
Group*Interval*Interval	P vs. E	-.02	.07	-0.34	.73
	M vs. E	.09	.07	1.23	.22
RD*Group*Interval	P vs. E	.15	.60	0.24	.81

	M vs. E	-.37	.71	-0.52	.60
RD*Group*Interval *Interval	P vs. E	-.01	.11	-0.10	.92
	M vs. E	.10	.14	0.68	.50

†p<.10 *p<.05 **p<.01 ***p<.001

Table 23

Group, interval, and RD effects in the prediction of PA, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.45	.08	5.60	<.001***
Residual		.25	.02	11.16	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		2.12	.28	7.60	<.001***
Between subject (*df* = 82)					
RD		.31	.65	0.47	.64
Group	P vs. M	-.77	.39	-1.98	.051†
Within subject (*df* = 249)					
Interval		.90	.23	3.92	<.001***
Interval*Interval		-.19	.05	-4.24	<.001***
Within & between subject interaction (*df* = 249)					
RD*Group	P vs. M	-.38	.80	-0.47	.64
RD*Interval		-.49	.53	-0.92	.36
RD*Interval*Interval		.10	.11	0.91	.36
Group*Interval	P vs. M	.51	.32	1.60	.11
Group*Interval*Interval	P vs. M	-.11	.06	-1.72	.09†
RD*Group*Interval	P vs. M	.51	.66	0.78	.44
RD*Group*Interval *Interval	P vs. M	-.11	.13	-0.82	.41

†p<.10 *p<.05 **p<.01 ***p<.001

Table 24

Group, interval, and testing effects in the prediction of PA, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.42	.06	6.70	<.001***
Residual		.27	.02	13.59	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		-.78	.80	-0.97	.33
Between subject (*df* = 125)					
Pre/Post		1.39	.50	2.78	.006**
Group	P vs. E	1.89	1.08	1.74	.08†
	M vs. E	2.50	1.11	2.25	.03*
Within subject (*df* = 367)					
Interval		2.80	.67	4.16	<.001***
Interval*Interval		-.54	.13	-4.06	<.001***
Within & between subject interaction (*df* = 367)					
Pre/Post*Group	P vs. E	-1.24	.67	-1.84	.07†
	M vs. E	-1.05	.72	-1.46	.15
Pre/Post*Interval		-.93	.42	-2.23	.03*
Pre/Post*Interval*Interval		.17	.08	2.08	.04*
Group*Interval	P vs. E	-1.77	.91	-1.94	.05†
	M vs. E	-1.80	.93	-1.94	.05†
Group*Interval*Interval	P vs. E	.34	.18	1.91	.06†
	M vs. E	.32	.18	1.73	.08†
Pre/Post*Group*Interval	P vs. E	1.18	.57	2.09	.04*

	M vs. E	.76	.60	1.27	.21
Pre/Post*Group*Interval *Interval	P vs. E	-.24	.11	-2.16	.03*
	M vs. E	-.13	.12	-1.09	.28

†$p<.10$ *$p<.05$ **$p<.01$ ***$p<.001$

Table 25

Group, interval, and testing effects in the prediction of PA, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.44	.08	5.64	<.001***
Residual		.25	.02	11.23	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		1.72	.74	2.32	.02*
Between subject (*df* = 85)					
Pre/Post		.33	.51	0.66	.51
Group	P vs. M	-.62	1.02	-0.60	.55
Within subject (*df* = 250)					
Interval		1.00	.61	1.63	.10
Interval*Interval		-.22	.12	-1.84	.07†
Within & between subject interaction (*df* = 250)					
Pre/Post*Group	P vs. M	-.19	.67	-0.28	.78
Pre/Post*Interval		-.16	.42	-0.39	.70
Pre/Post*Interval*Interval		.04	.08	0.49	.63
Group*Interval	P vs. M	.02	.85	0.03	.98
Group*Interval*Interval	P vs. M	.03	.17	0.17	.87
Pre/Post*Group*Interval	P vs. M	.42	.56	0.75	.46
Pre/Post*Group*Interval *Interval	P vs. M	-.11	.11	-1.01	.32

†p<.10 *p<.05 **p<.01 ***p<.001

Table 26

Group and interval effects in the prediction of PA excluding patients who had pre lab, P vs. E & M vs. E contrasts

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.43	.09	4.79	<.001***
Residual		.24	.02	9.52	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		1.99	.32	6.21	<.001***
Between subject (*df* = 60)					
Group	P vs. E	-.59	.44	-1.34	.18
	M vs. E	.40	.50	0.80	.43
Within subject (*df* = 181)					
Interval		.94	.26	3.59	<.001***
Interval*Interval		-.20	.05	-3.84	<.001***
Within & between subject interaction (*df* =181)					
Group*Interval	P vs. E	.58	.36	1.62	.11
	M vs. E	-.27	.42	-0.65	.52
Group*Interval*Interval	P vs. E	-.14	.07	-1.93	.06†
	M vs. E	.06	.08	0.69	.49

†p<.10 *p<.05 **p<.01 ***p<.001

Table 27

Group and interval effects in the prediction of PA excluding patients who had pre lab, P vs. M contrast

Random effects					
Covariance parameter estimate		β	SE	*Z*	*p*
UN (1,1)		.44	.11	3.85	<.001***
Residual		.25	.03	7.65	<.001***
Fixed effects					
Predictor variable	Contrast	β	SE	*t*	*p*
Intercept		2.39	.40	6.95	<.001***
Between subject (*df* = 39)					
Group	P vs. M	-.99	.51	-1.95	.06†
Within subject (*df* = 117)					
Interval		.68	.33	2.03	.045*
Interval*Interval		-.14	.07	-2.16	.03*
Within & between subject interaction (*df* = 117)					
Group*Interval	P vs. M	.85	.42	2.03	.04*
Group*Interval*Interval	P vs. M	-.19	.08	-2.33	.02*

†p<.10 *p<.05 **p<.01 ***p<.001

Figure 1

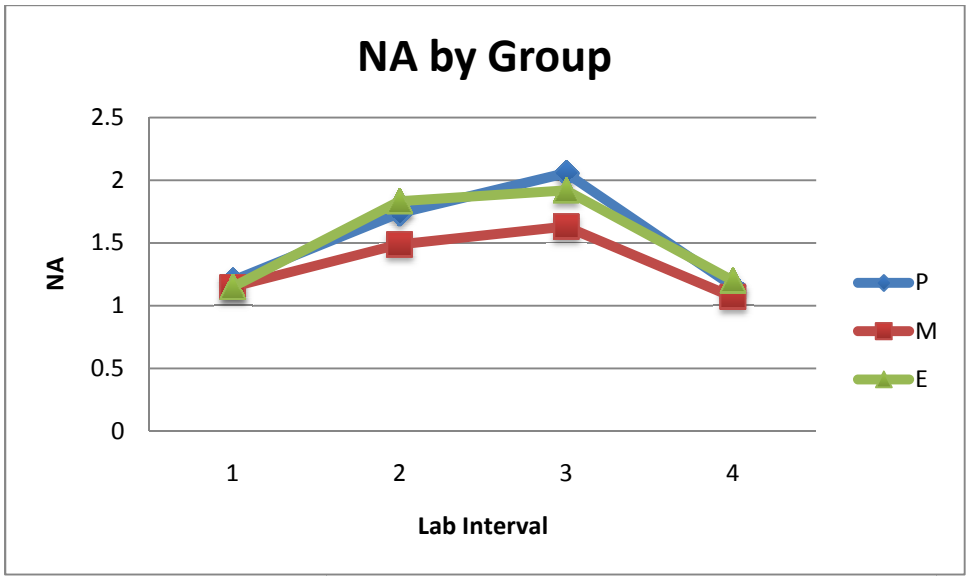


Figure 2

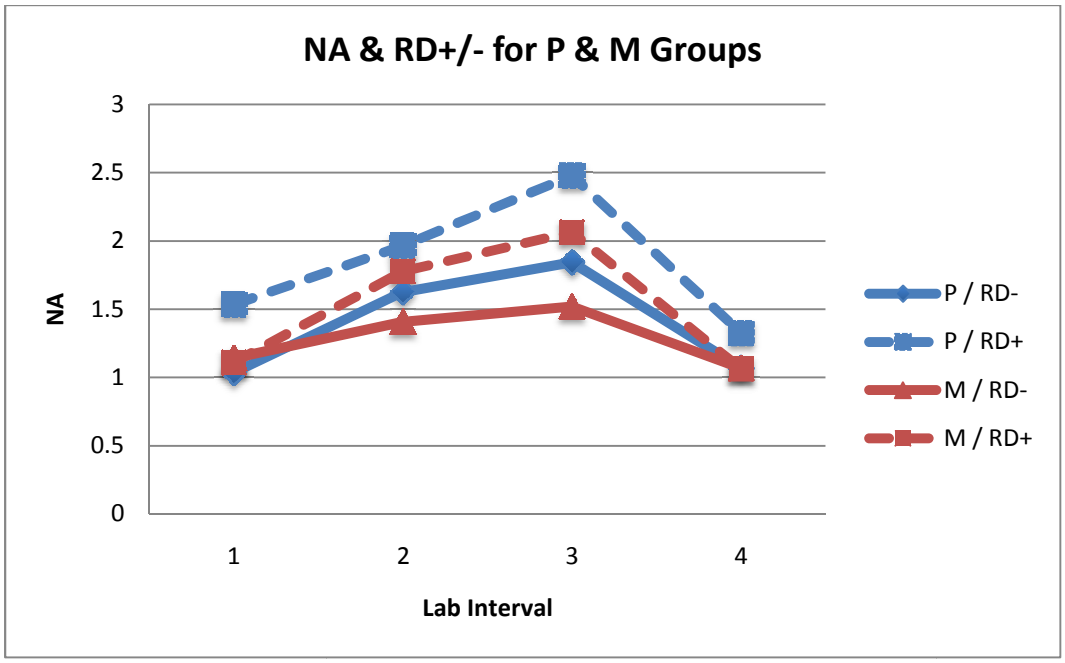

NA & RD+/- for P & M Groups
NA
3
2.5
2
1.5
1
0.5
0
1
2
3
4
Lab Interval
P / RD-
P / RD+
M / RD-
M / RD+

Figure 3

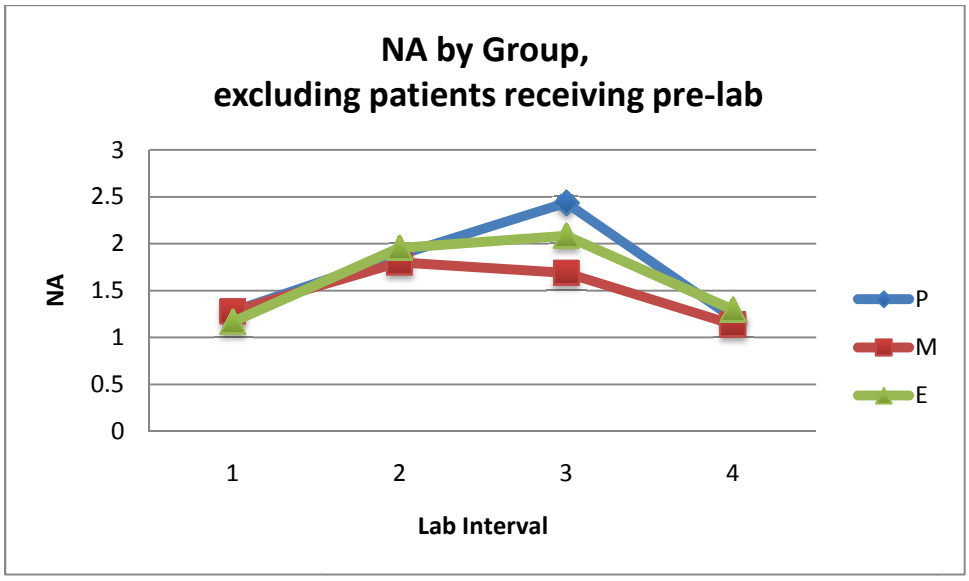

NA by Group,
excluding patients receiving pre-lab
NA
3
2.5
2
1.5
1
0.5
0
1
2
3
4
Lab Interval
P
M
E

Figure 4

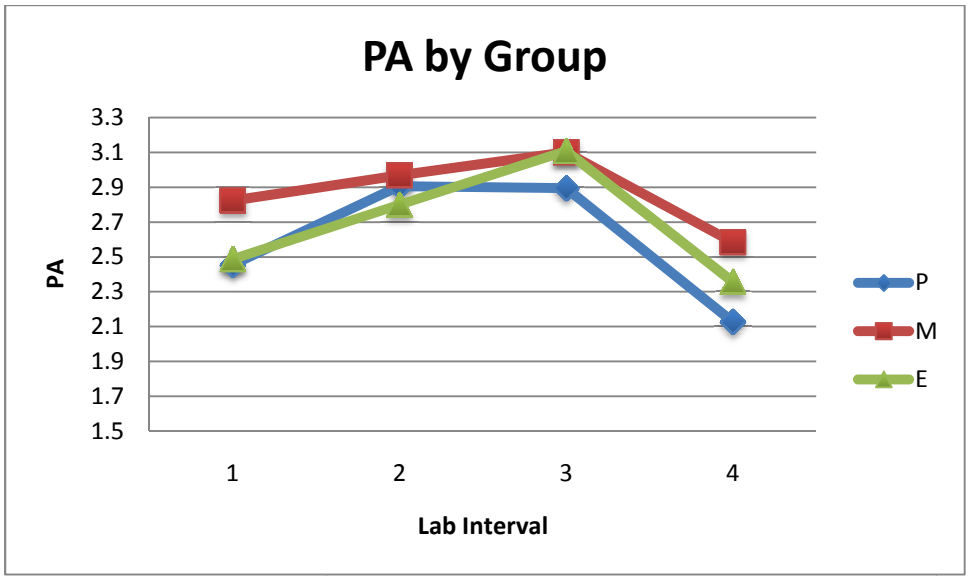
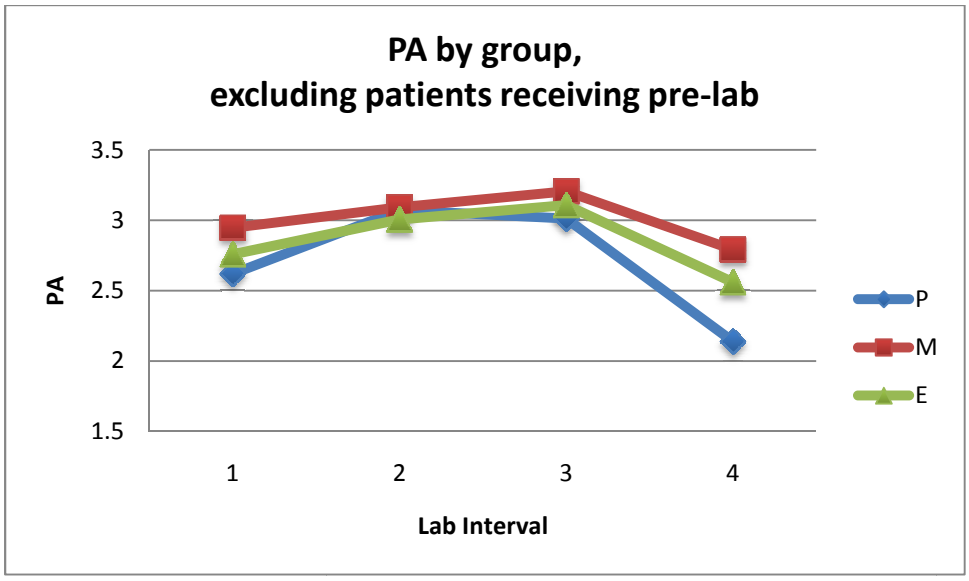


Figure 5


PA by group,
excluding patients receiving pre-lab
3.5
3
2.5
2
1.5
PA
1
2
3
4
Lab Interval
P
M
E